REPLY

We appreciate the kind comments and provocative suggestions of Kereiakes et al. We particularly relish the appellation "bell weather" (*sic*). (While "Bellwether" connotes leadership, we hope the writers did not intend the term's more literal Old English usage: a gelded male sheep wearing a bell leading a foolish flock!) (1).

In our manuscript (2), we did not intend to propose detailed primary angioplasty guidelines nor formal protocols for general use at nonsurgical hospitals. Such a project would require much input from numerous sources. We do agree with Kereiakes et al. that experienced, high-volume operators are essential to the success of such programs, as are an experienced support team (see paragraph 5 below) and formalized transfer protocols.

We do not believe that reports of less favorable outcomes of coronary intervention at lower-volume hospitals should be a reason to limit *primary* angioplasty to higher-volume hospitals, for multiple reasons:

1. The reported differences in mortality for elective angioplasty between the lowest- and highest-volume hospitals are very small. A recent American College of Cardiology (ACC) clinical competence statement on coronary intervention (3) included reviews of 11 papers that related institutional interventional volume to outcomes. Only 3 of these 11 (4–6) found any relation between institutional volume and mortality in hospitals in the early 1990s performing over 25 to 50 cases a year; these differences in absolute mortality were only 0.2% to 0.8%. Five of the 11 studies examined data on patients with acute myocardial infarction (AMI) (7–11); none of these five found any volume-related mortality differences in hospitals performing over 40 to 50 cases annually.
2. Lower-volume hospitals may perform a disproportionately larger percentage of interventions on patients with high-risk diagnoses such as AMI. Most of the above reports do not use risk-adjusted data, and thus they may have "disregarded robust risk factors that explain most of the variation in outcome attributed to hospital volume" (12).
3. All else being equal, AMI patients *might* have a very slight advantage if they were to present to a higher-volume rather than a lower-volume hospital—if each facility practiced primary angioplasty routinely. But all else is not equal. We must compare the outcomes of primary angioplasty at lower-volume hospitals, *not* to outcomes at higher-volume hospitals, but rather to the risk of not offering reperfusion therapy at all to the majority of patients who are lytic-ineligible, or to the risk and delay of transfer of such patients to a willing surgical center for primary angioplasty. The differences in mortality among these alternatives may be far greater than the ≤0.8% cited above. As yet, there is no "evidence-based" reason to conclude that either of these two alternatives is superior to early primary angioplasty on-site. Unfortunately, randomized studies of such issues have met with only limited success in enrolling patients.
4. Outcomes data on low-volume hospitals may disproportionately reflect the outcomes of low-volume operators. Outcomes of high-volume operators at low-volume institutions have not been examined. We propose that primary angioplasty be performed at such hospitals only by higher-volume operators who also routinely perform elective angioplasty at surgical centers.
5. We agree that primary angioplasty is different from elective angioplasty and that it requires simultaneous intensive medical care of the acutely ill AMI patient. Relatively more patients with AMI present to community hospitals than to tertiary centers. Thus, the nursing and technical staff at smaller hospitals may already have thorough experience in the medical care of such patients. Further, smaller hospitals with only one catheterization team and few operators may gain more experience in performing primary angioplasty—on a per operator and per team basis—than larger surgical centers with many more teams and operators. (Many high-volume hospitals and operators perform little if any primary angioplasty, having full schedules that do not easily permit interruptions).
6. Available evidence from many reported series besides our own indicates that qualified hospitals without cardiac surgery can achieve primary angioplasty that are similar to those of high-volume surgical centers (13–23).
7. In the ACC clinical competence statement (3), Hirshfield et al. note that the studies cited therein were before the era of stents and platelet GP IIb/IIIa inhibitors, and they observe, "It is likely that the availability of these treatments has reduced the expected frequency of death and emergency CABG. . . . Consequently, these data may not accurately reflect current practice." Along these same lines, Teirstein (24) editorializes that "the dramatically low event rate [with stents and newer antiplatelet regimens] begs the question: Is elective coronary stenting now so predictable that outcomes are not longer operator dependent?" Teirstein goes on to add, "Recent data support the use of a direct mechanical approach to acute infection. However, widespread acceptance of this technique will require increased patient access to adequate physician and institutional expertise. . . . Therefore, to achieve overall public health benefits, *credentialing for these urgent procedures may necessarily be different from elective procedures*" [italics added].

We agree with Kereiakes et al. that launching a primary angioplasty program may be a daunting challenge, both logistically and economically, at small hospitals. This challenge has been successfully met by over 50 hospitals without cardiac surgery in the U.S. (15–22; personal survey by Wharton) and by large numbers of hospitals around the globe (11,13,14,23). Regarding the "cost consequences," primary angioplasty may be more cost-effective than fibrinolytic therapy (25–28). The addition of stents and platelet GP IIb/IIIa inhibitors, while greatly enhancing the safety of primary angioplasty, may also further enhance its cost-effectiveness (29–33).

We disagree that newer pharmacology is likely to render primary angioplasty "obsolete." Two-thirds of patients with AMI are not candidates for fibrinolytic therapy, either because they have bleeding risks, shock, present late, or do not have diagnostic electrocardiograms; these patients are generally at higher risk than fibrinolytic-eligible patients, and they need a reperfusion alternative to "morphine and bedrest" (34–37). Further, the citation by Kereiakes et al. of a 70% reperfusion rate at 60 min for patients treated with combination fibrinolytic and platelet GP IIb/IIIa inhibitor therapy is based on very small numbers of patients in two pilot studies (38,39) and thus is not yet thoroughly "evidence-based."

Kereiakes and colleagues cite the time delay for primary angioplasty as a disadvantage. Time delay, of course, is a factor in both higher- and lower-volume hospitals. Regardless, the outcomes of primary angioplasty are better than those of fibrinolytic therapy (40) and are not nearly so time-dependent (41). In fact, if the

average "door-to-needle" time is as long as 45 min (10) and the average "needle-to-reperfusion" time is 60 to 90 min (42), then the average time-to-reperfusion for the small majority of fibrinolytic-treated patients who do achieve TIMI flow grade 3 (43) is very similar to the time-delay for on-site primary angioplasty. The latter treatment results in reperfusion in about 95% of patients (44–50) and can be applied to a much greater proportion of patients with AMI at capable centers.

Offering effective primary angioplasty at the point of first contact can provide the most rapid reperfusion alternative for the two-thirds of patients with AMI who are not lytic-eligible. This may be particularly important for patients in cardiogenic shock. In the SHOCK study (51), revascularization in under 6 h after onset of AMI conferred the greatest survival advantage of all descriptors examined. However, universal triage of AMI patients to high-volume hospitals could quickly flood the capability of their catheterization laboratories, which often are already working at full capacity. One possible solution is the establishment of specialized "Emergency Heart Attack Treatment Centers" with around-the-clock primary angioplasty capability (though not necessarily with on-site cardiac surgery) to which all patients with AMI could be triaged by ambulance.

We agree that updated guidelines are now needed that will more fully address the performance of primary angioplasty at nonsurgical hospitals. Input from physicians who practice in these circumstances should be sought. It is now not only possible, but in fact imperative, to offer this potentially life-saving therapy to more patients with AMI in broader geographical locations.

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Nonocclusive Coronary Dissections: To Stent or Not to Stent?

Cappelletti et al. (1) reviewed the outcome of 129 consecutive patients treated with conventional angioplasty (PTCA) at a time when coronary stents were not available. Patients (45; 35%) presenting nonocclusive dissections post-PTCA had a significantly lower restenosis rate than patients without dissections (12% vs. 44%; $p < 0.001$). The restenosis rate in another group of patients undergoing stenting for nonocclusive dissection (clinically and angiographically matched), later on in their experience, was 25% (1). At a time when coronary stenting is experiencing an exponential increase, these results would appear rather provocative. Some classical studies, however, also suggested that most dissections are not only benign but also predict a lower restenosis rate (2,3). Nevertheless, no previous study was able to demonstrate such a low restenosis rate in this cohort of patients.

Furthermore, to demonstrate convincingly that a conservative approach—namely a "watchful waiting" strategy—may even be superior in the long run to coronary stenting is much more challenging. Given the potential clinical implications of this study, some methodological clarifications appear warranted.

First, it is not clear why two patients with vessel closure were excluded. Keeping in mind that this is a retrospective study, it will be important to know whether these dissections were flow-limiting immediately after PTCA or flow deterioration occurred later on. Second, 67% (33/49) of the nontreated dissections were type A versus none (0/60) of the stented dissections (chi-square $p < 0.0001$). Therefore, it is difficult to assume that these two populations were similar, and thus direct comparison of results may not be appropriate. Further details on whether the restenosis rate tended to cluster around patients with type C-D dissections (untreated/stented groups) will be helpful.

Finally, the methodology of quantitative coronary analysis was not specified. This is relevant because the analysis of dissected coronary segments is technically demanding. In fact, at first glance it appears difficult to explain a mean lumen diameter post-PTCA of 3.23 ± 0.65 mm (reference 3.20 ± 0.54 mm) yielding a $20 \pm 7\%$ diameter stenosis. The large lumen diameter of the dissected segments indicates that the dissection image was fully included into the lumen measurements. This is in contradistinction with some prior studies using careful edge-detection quantitative angiography (4,5). We previously demonstrated (5) that residual coronary dissections after stenting had a benign outcome when they were stable, were not associated with significant lumen narrowing, and did not compromise coronary flow.

Our data (5) also concur with the current study, suggesting that most residual dissections disappear at follow-up. Moreover, these dissected coronary segments may promote a unique pattern of vessel remodeling that could explain a lower restenosis rate (1) or even a significant lumen improvement on late angiography (5). We fully agree with the idea that conservative management of coronary